maffin content of the adrenals while others, such as sodium cacodylate, salvarsan, and neosalvarsan, cause a rapid and marked decrease in this substance, as judged by the color of the medullary cells after fixation in Müller's fluid.

From these observations it appears that the adrenotropic action of arsenicals is one of the most constant and important features of arsenical intoxication and we suggest that therapeutic doses of some arsenicals may be found to produce definite stimulation of the adrenal glands.

VARIATIONS IN THE CHARACTER AND DISTRIBUTION OF THE RENAL LESIONS PRODUCED BY COMPOUNDS OF ARSENIC

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The chemical agents employed in the production of experimental nephritis are usually divided into two classes, those producing tubular lesions and those producing vascular lesions, to the latter group of which the compounds of arsenic have been assigned. From a study of the renal lesions produced by a large number of arsenicals, however, we have been led to question the validity of such a classification and to view the pathogenic action of such substances from the standpoint of their chemical constitution as well as their arsenic content.

The classical hemorrhagic kidney of arsenious acid is by no means constant for all compounds of arsenic. Grossly, the kidneys of dogs given lethal doses of such substances as arsenious acid, salvarsan, neosalvarsan, galyl, arsenophenylglycine, atoxyl, and arsacetin are separable into two extreme groups, the red and the pale kidneys, with transitional types in which the predominating changes ally them more closely with the one group or the other. In the group of red kidneys, congestion and hemorrhage are the dominant features of the arsenical action, while in the pale kidneys, the dominant lesion is tubular.

Upon closer analysis of the gross and microscopic changes we can make a further differentiation of the action of compounds that produce kidneys of the one or the other of these types. For example, arsenious acid, salvarsan, neosalvarsan, and galyl all produce red kidneys, but the congestion and hemorrhage produced by arsenious acid is diffuse in character with but slight tubular necrosis, while the vascular injury of salvarsan, neosalvarsan, and galyl is more pronounced in the cortex and the boundary zone and is accompanied by much more marked

tubular necrosis and interstitial edema. Again, atoxyl causes extensive hemorrhage extending from the boundary zone through the medulla and only slightly into the cortex, the outer rim of which remains pale; there is marked degeneration and necrosis of tubular epithelium. These atoxyl kidneys which are outwardly pale combine in a peculiar form the essential features of both the red and the pale kidneys. In like manner, arsenophenylglycine produces kidney lesions of a combined type and while tubular degeneration and necrosis are dominant there is usually some congestion and hemorrhage in the boundary zone and medulla—more rarely in the cortex.

The other extreme in the action of arsenicals upon the kidney is exemplified by arsacetin which produces a typically pale kidney. While congestion and hemorrhage are still apparent to a minor degree in the boundary zone of these kidneys the vascular injury is so completely overshadowed by the injury to the tubular epithelium as to leave no doubt as to the dominance of tubular injury. Further, the prompt and vigorous regeneration of the tubular epithelium indicates that the extensive necrosis produced by arsacetin can not be regarded as a secondary anemic phenomenon.

It is certain, therefore, that all arsenicals do not produce renal lesions that are identical either in character or distribution but that this group of substances includes agents producing a so-called tubular nephritis as well as those producing a vascular nephritis, and that these wide differences in the pathogenic action of different compounds of arsenic are explainable only upon the basis of their chemical constitution.

SEVEN POINTS ON A TWISTED CUBIC CURVE

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Six points in space, barring special situations, determine a twisted cubic curve. From any seventh point of the curve those six are projected by six generators of a quadric cone. For any seven points of a cubic curve there is accordingly a symmetric set of seven cones; and it is well known that seven points giving rise to two such cones are on a cubic curve, and so give rise to five more cones. This is the only current theorem on seven points of a twisted cubic. Concerning eight points there is the elegant theorem of von Staudt, that if two tetraedrons have eight points of a twisted cubic for vertices, their eight faces osculate a second cubic curve. I propose to demonstrate a theorem whose formulation resembles the latter, while like the former it relates to the